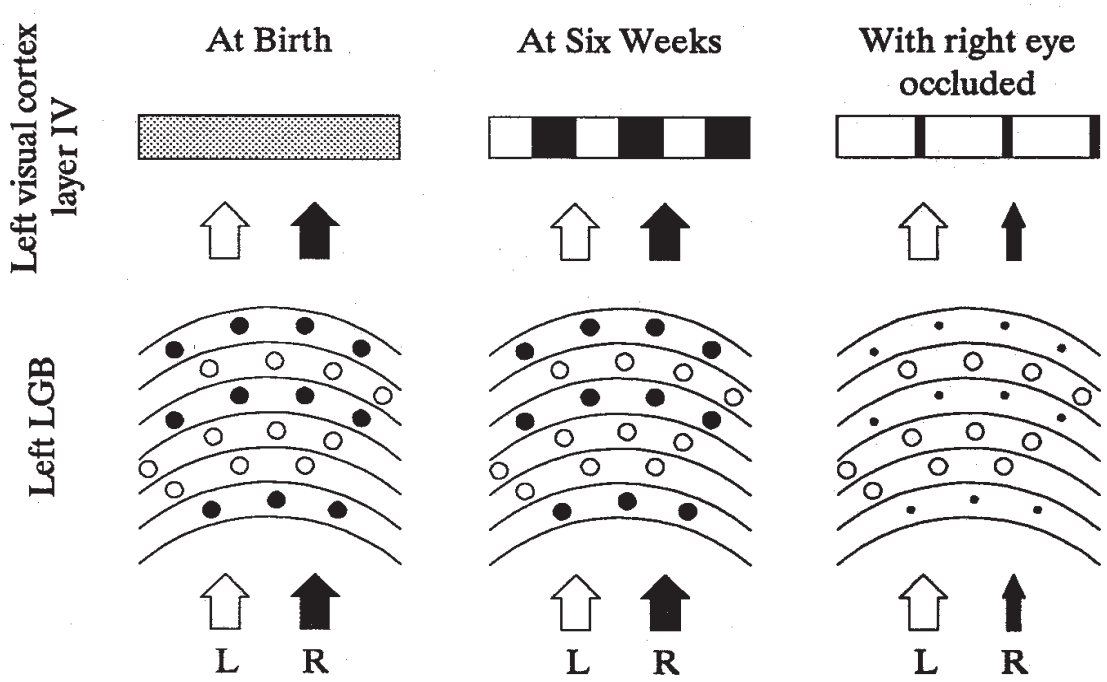
SENSORY PLASTICITY

You may have the idea that the visual, auditory and somatosensory systems are static pathways (i.e., the neural wiring is in place and simply does its job). To some extent, this is true, but there is also an important dynamic component to sensory systems. Sensory systems exhibit **plasticity**, *the capacity to make functionally appropriate adjustments in neural connection patterns*. Sensory systems are not mature at birth. Considerable modification takes place postnatally. Also, some reorganization within sensory structures and "rewiring" of neural connections can occur in the sensory pathways in adults. This restructuring is part of normal processes and may also be an important mechanism for recovery from injury.

A. PLASTICITY DURING DEVELOPMENT

Considerable neural development occurs postnatally in humans, and this is evident behaviorally. For example, infants at birth are legally blind by adult visual acuity standards, with incompletely developed color vision, 3-D stereovision, and eye movement control. By 8 months of age, vision approaches normal adult levels.



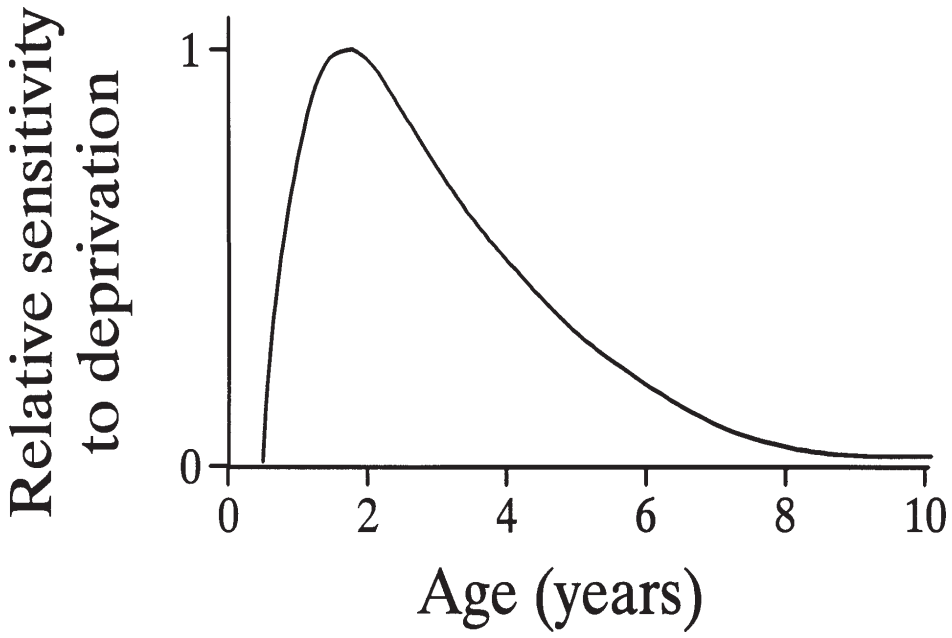
Normal development and effects of monocular deprivation in the visual system of the macaque monkey. Cells within a given layer of the LGB are innervated by a single eye. A: At birth, the projection from the two eyes to layer IV of area 17 is intermixed. B: The inputs from the two eyes segregate by six weeks, producing the alternating strips of ocular dominance columns. C: Without normal visual stimulation in the right eye, LGB cells shrink and cortical representation is substantially reduced. LGB, lateral geniculate body of the thalamus. L, left. R, right. From Daw (1987).

During infancy, the connections between sensory neurons continue to become established, refined, and removed. This is illustrated nicely by the emergence of normal ocular dominance columns in the monkey visual cortex and their disruption by visual deprivation. At birth, input from

the two eyes is intermixed in layer 4 of visual cortex, the primary layer receiving LGB innervation. Normally, by six weeks, the input is segregated into alternating, equal-sized columns of input from each eye. These columns of alternating ocular dominance resemble those in the adult. However, if the input from one eye is significantly compromised (e.g., by a long period of occlusion), then the cortex becomes dominated by input from the "good" eye, and the representation in the cortex from the "deprived" eye is reduced. This has long-term perceptual consequences, as vision from the deprived eye may remain poor, even if normal visual input is restored.

This is a very clear example of how the sensory connections that become established are influenced by sensory experience. Sensory pathways that are well used firmly establish their neural connections within central sensory structures. In contrast, sensory pathways that are little or un-used can become weakened and lose representation in central sensory structures.

Normal sensory experience is necessary for normal development. An important component in the development of sensory systems is the concept of a sensitive or **critical period**. The critical period is the time during development when neural connections are forming and the system is most vulnerable to the damaging effects of abnormal sensory input. In the visual system, faulty visual input, such as from a central cataract or strabismus, if not corrected before the end of the critical period, can result in permanent visual deficits, including functional blindness. Once a child is beyond the critical period, the visual deficit can remain permanent, even if the original source of the abnormal visual experience is corrected.



Time course of the sensitive period for susceptibility to visual deprivation in humans.
From Moses and Hart (1987).

The critical period in visual development involves the first six years of life, although the *first two or three years are most crucial*. The critical period in audition is less well defined, but appears to last longer.

Obviously, permanent compromise of sensory systems is not in an individual's best interest. Sensory deprivation can severely impact not only the afferent sensory pathway itself, but other cognitive functions, as well. For example, in the auditory system of infants, discrimination of speech sounds begins within a few hours or days of birth, and much of the perceptual basis for speech has been laid out by the 12th month after birth. Even a partial hearing loss during the early years of life can seriously damage speech production and the acquisition of language.

Thus, when sensory deficits are suspected, early intervention is critical to ensure the best possible outcome. It is valuable to recognize that vision and hearing assessments can be performed even in very young children and infants.

This is only meant to be a superficial introduction to the topic of neural plasticity during development. (Don't get bogged down in the experimental details of ocular dominance columns.) Focus on the fundamental concepts:

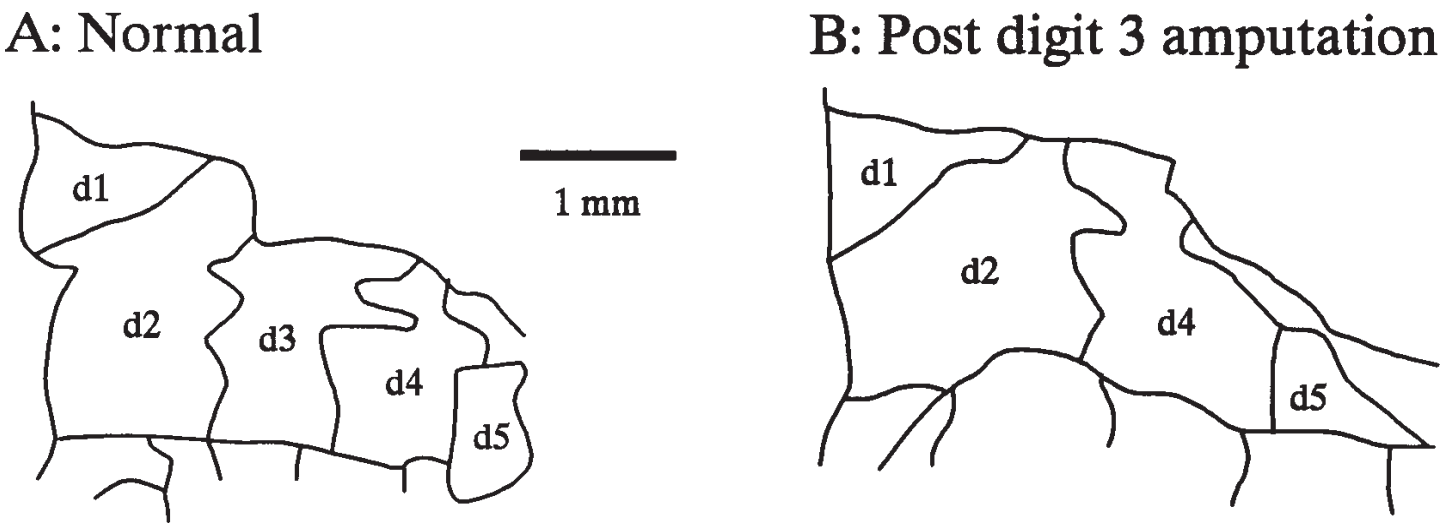
- That is, the sensory systems are immature at birth.
- The normal, postnatal development of our senseory systems depends on normal sensory experience.
- Sensory deprivation or abnormal sensory experience during the critical period (in early childhood), if uncorrected, can lead to permanent deficits.

B. PLASTICITY IN ADULTHOOD

Until comparatively recently it was thought that once sensory nerve pathways were established, no further restructuring could occur. However, it is now apparent that sensory neural reorganization can take place in adults under a variety of circumstances.

Reorganization After Peripheral Lesions

Sensory reorganization was first demonstrated in the adult somatosensory cortex following peripheral nerve lesions. It was thought that such a lesion would result in a "silent zone" in the corresponding region of somatosensory cortex, since those previously innervated cortical neurons would be deprived of their somatosensory input. However, detailed somatotopic mapping of cortical areas before and after peripheral denervation shows that the "sensory deprived" neurons do not become silent. Instead, they become responsive to adjacent skin territories with intact peripheral innervation. The cortical map shifts as adjacent subregions functionally take over and fill in the denervated zone.



Plasticity in cerebral cortical area 3b (in S1) of an adult owl monkey. A: Normal somatotopic map of the digits of the hand. B: Somatotopic map of the same region shown in part A, obtained two months after the third digit was amputated. From Merzenich, et al. (1984).

Although extensive reorganization has been observed to occur over several months, post denervation, some of these changes can occur within minutes of the lesion. This plasticity has also been reported in VPL, the dorsal column nuclei in the medulla and the dorsal horn in the spinal cord. Similar results are reported in the visual system.

Reorganization After Central Lesions

Sensory plasticity in the adult has also been observed following lesion in the cerebral cortex. In this case, the peripheral innervation is intact, but the central nervous system target is not. The cortical reorganization shifts so that the somatotopic location that was mapped originally onto the lesioned cortical region is now mapped adjacent to the lesion. These results suggest one mechanism for recovery from stroke.

Plasticity With Learning

Finally, an expansion of a particular somatotopic area of the cerebral cortex is also observed in association with learning. For example, somatotopic reorganization was observed in adult monkeys who were trained on a tactile discrimination task involving exclusively the use of a particular digit. Following the training, the cortical representation of that digit had expanded significantly.

Clearly, we must not think of the brain as a completely hard-wired series of connections between neurons. Instead, it is a structure that can adapt to the sensory environment and rearrange to meet the needs of the body.